to about 130% of the level present in erythrocytes of nonpregnant women.[2] In this study, the actual and the standardized P 50 were increased to about 2,5–3,0 mmHg. Moreover, hormonal changes during pregnancy could affect the red cell metabolism, *e.g.*, the 2,3 DPG level has been shown to be sensitive to changes in the concentration of thyroxin and prostaglandin.[2,3]

Furthermore, the authors did not discuss the leftward shift of the oxyhemoglobin dissociation curve in preeclamptic parturients. One explanation for the leftward shift could be hypophosphatemia which increases oxygen affinity of hemoglobin.[4] This electrolyte imbalance could be induced by excessive loss of phosphate, which occurs when hypokalemia is provoked by diuretic therapy in these patients. In any case, this remarkable observation should be commented upon.

WOLFGANG HESS
Assistant Professor of Anesthesiology
Department of Anesthesiology
University of Essen
Hufelandstr. 55
4300 Essen 1 Federal Republic of Germany

REFERENCES

1. Kambam JR, Handte RE, Brown WU, Smith BE: Effect of normal and preeclamptic pregnancies on the oxyhemoglobin dissociation curve. ANESTHESIOLOGY 65:426–427, 1986
2. Rorth M, Brake NE: 2,3 DPG in human pregnancy, Oxygen affinity of Hemoglobin and Red Cell Acid Base Status. Edited by Rorth M, Astrup P. New York, Academic Press, 1971, pp 692–695.
3. Synder LM, Reddy WJ: Mechanism of action of thyroid hormones on erythrocyte 2,3-diphosphoglyceric acid synthesis. J Clin Invest 49:1993–1998, 1970
4. Sladen RN: The oxyhemoglobin dissociation curve, Clinical Aspects of Oxygen. Edited by Brodsky JB. Boston, Little Brown and Company 1981, pp 39–70

(Accepted for publication February 24, 1987.)

Anesthesiology
66:855, 1987

In Reply:—The effect of normal pregnancy on oxyhemoglobin dissociation (OHD) has been reported by several investigators. A leftward shift of OHD seen in preeclamptic pregnant women is new and surprising. Both normal and preeclamptic pregnancies are associated with complex changes in a variety of hormones, including prostaglandins. Several of these hormones are also known to cause shifts in OHD. We have discussed the possible causes for a leftward shift of OHD in a recent abstract describing the effect of pregnancy induced hypertension on OHD.[1] Evidence also suggests that there is increased red cell destruction in patients with toxemia of pregnancy.[2] In addition, we have recently reported that preeclamptic patients have significantly higher levels of carboxyhemoglobin compared with normal pregnant women.[3] We believe that the increased level of carboxyhemoglobin is principally responsible for the observed leftward shift in OHD. Our preeclamptic patients did not receive any diuretic therapy and, thus, hypophospatemia from diuretic therapy is unlikely. Also, none of our patients at the time of the study was receiving any other drug that could possibly cause a shift in OHD.

J. REDDY KAMBAM, M.D.
Assistant Professor of Anesthesiology
Department of Anesthesiology
Vanderbilt University
Nashville, Tennessee 37232

REFERENCES

1. Kambam JR, Handte RE, Brown WU, Smith BE: Effect of pregnancy induced hypertension on oxyhemoglobin dissociation. ANESTHESIOLOGY 59:A396, 1983
2. Entman S, Richardson LD, Killam AP: Altered ferrokinetics in toxemia of pregnancy. A possible indicator of decreased red cell survival. Clin Exp Hypertens B2: 171–178, 1983
3. Kambam JR, Entman S, Mouton S, Linn J, Smith BE: Effect of preeclampsia (PIH) on carboxyhemoglobin levels: A mechanism for shift of P50 to the left (Abstract) ANESTHESIOLOGY 65: A393, 1986

(Accepted for publication February 24, 1987.)

Anesthesiology
66:855–856, 1987

*p*H-adjusted Lidocaine Does Not "Sting"

To the Editor:—The widespread practice at our institution of adding sodium bicarbonate to lidocaine solutions (ratio 1:10) to improve the success rate of regional anesthesia[1] has led to the observation that these *p*H-adjusted solutions do not "sting" when injected intradermally or subcutaneously. The improvement in comfort when providing cutaneous anesthesia is striking. Starting an iv in a child, for example, is greatly facilitated when *p*H-ad-